specific putative receptor molecule of the immunoglobulin superfamily. Analyses of Tutl PNS expression revealed localization to Class III and IV dendritic arborization (da) neurons, suggesting that *tutl* may regulate class-specific dendritogenesis. Loss-of-function (LOF) analyses revealed cell-autonomous functions for *tutl* in promoting Class III and IV dendritic arborization and receptive field innervation. Conversely, gain-of-function (GOF) studies revealed ectopic Tutl expression in Class I da neurons results in increased dendritic complexity. The *tutl* LOF and GOF phenotypes, as well as Tutl PNS expression patterns, are similar to those observed for the Cut homeodomain transcription factor, recently demonstrated to mediate class-specific da neuron dendritogenesis. We therefore examined the potential that Cut may transcriptionally regulate Tutl expression in da neurons. Both LOF and GOF cut analyses suggest Tutl represents the first known downstream Cut transcriptional target in the regulation of class-specific da neuron dendrite morphogenesis.

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Program/Abstract # 409

The zebrafish calpain system – expression and role of calpain and calpastatin during early development

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The calpain superfamily is a large group of highly conserved calcium-dependent cysteine proteases that have been implicated in regulating a wide variety of biological processes such as cell adhesion, migration and intracellular signaling. Two heterodimeric typical family members, μ-calpain and m-calpain, have been studied extensively in vitro and in cell culture but few studies have been aimed at determining the function of calpain and its endogenous inhibitor calpastatin in vivo. Recently, calpain knockout mice have revealed that both m-calpain subunits are indispensable for survival of the pre-implantation embryo; however, the precise role the calpain system plays during early development has yet to be determined. We have cloned and characterized the temporal and spatial expression of four zebrafish genes encoding typical calpain catalytic subunits (capn1a, 1b, 2a, 2b), two genes encoding common regulatory subunits (cpns1a, 1b) and calpastatin (cast). RT–PCR and whole-mount in situ hybridization analysis reveals that these genes are expressed in distinct, yet overlapping, spatiotemporal patterns during the first 24 h of development. Preliminary loss-of-function experiments, employing chemical calpain inhibitors, cpns-directed morpholinos and calpastatin over-expression, suggest the calpain system might be necessary for the successful completion of morphogenetic movements, such as epiboly, and proper patterning of the dorsal–ventral axis.

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Crip2 has dual functions in the cytoplasm and nucleus, induces non-canonical Wnt signaling during convergent extension movement in zebrafish notochord

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In notochord development, ntl induce intercalation of the cells, but their target or signaling cross-talks were unclear. We performed microarray experiment using ntl knockdown embryo of zebrafish and identified *Cysteine-rich protein 2* (*crip2*) as a transcriptional target of ntl. Crip2 expressed specifically in the notochord and regulate convergent extension cell movement of gastrulation. By molecular and cellular assay, Crip2 was localized in the nucleus on Wnt stimulation. In the nucleus, Crip2 bind to β-catenin and inhibited β-catenin/Tcf-dependent transcription. Moreover, Crip2 was also localized in cytoplasm, directly interacted with Dishevelled 2 and formed a complex with Crip2/Dvl2/Daam1/Diversin on non-canonical Wnt stimulation. Moreover Crip2 recruited this complex to the focal adhesion complex near the leading edge of cell and control cell morphology and migration. This is a first report elucidating the molecular mechanism of intercalation and directly interactions between ntl and canonical and non-canonical Wnt signaling.

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Program/Abstract # 411

Cadherin-based adhesion cooperates with non-canonical Wnt signaling to mediate morphogenesis in the zebrafish

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A critical step in central nervous system development is the shaping of the neural tube (or neurulation). *N-cadherin* (*N-cad*), a calcium-dependent, homophilic-binding cell adhesion molecule, has a conserved role in this process in vertebrates as disruption of *N-cad* results in a variety of neural tube defects. However, the role of *N-cad* in regulating the cellular and molecular mechanisms underlying neurulation has not been clearly elucidated. By direct analysis of cell behaviors, we have shown that although *N-cad*-depleted cells are not defective in their ability to form protrusions, they are not able to maintain them stably. Here, we begin to address whether *N-cad* functions solely as an adhesion molecule